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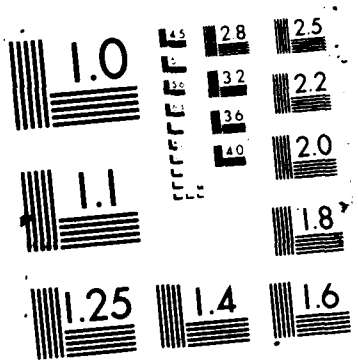
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Menstrual Cycle Effect on the Control of Skin Blood Flow during Exercise at High Core Temperatures

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(U) Skin blood flow (SkBF) is linearly related to core temperature (T_c) during exercise. A break point occurs in the SkBF:T_c relationship during exercise such that the increase in SkBF per unit increase in T_c is attenuated in men at a T_c of approximately 38°C. Five women were studied during exercise (35 min) at 80% $\dot{V}O_2$ peak (T_a = 35°C; T_{dp} = 10° C) during the mid-follicular phase and mid-luteal phase of the menstrual cycle to determine if the onset of this vasoconstrictor activity is affected by the elevated luteal T_c. T_c was measured every 30 s by a thermocouple placed in the esophagus. SkBF was measured twice per min by venous occlusion plethysmography. T_c and SkBF were calculated for that point. The onset of vasoconstrictor activity occurred at 37.53(±0.1)°C and 10.9(±2.4) ml·100 ml^{-1}·min^{-1} in the mid-follicular phase and at 38.20(±0.2)°C and 14.6(±2.2) ml·100 ml^{-1}·min^{-1} in the mid-luteal phase. Both T_c and SkBF were significantly increased at the onset of vasoconstrictor activity in the luteal phase ($p \leq 0.05$). Heart rate averaged 150 (±11) and 161 (±9) beats·min^{-1} at the break point during the follicular and luteal phases, respectively. These

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data indicate that onset of vasoconstrictor activity occurred at an increased T_c and SkBF during the luteal than in the follicular phase and therefore is not dependent on a critical exercise SkBF or T_c but may be explained by another regulatory mechanism, perhaps arterial pressure regulation.

Skin blood flow (SkBF) is linearly related to core temperature (T_c) during exercise. A break point occurs in the SkBF:T_c relationship during exercise such that the increase in SkBF per unit increase in T_c is attenuated in men at a T_c of approximately 38°C. Five women were studied during exercise (35 min) at 80% $\dot{V}O_2$ peak (T_a = 35°C; T_{dp} = 10°C) during the mid-follicular phase and mid-luteal phase of the menstrual cycle to determine if the onset of this vasoconstrictor activity is affected by the elevated luteal T_c. T_c was measured every 30 s by a thermocouple placed in the esophagus. SkBF was measured twice per min by venous occlusion plethysmography. T_c and SkBF were calculated for that point. The onset of vasoconstrictor activity occurred at 37.53(±0.1)°C and 10.9(±2.4) ml•100 ml^{-1}•min^{-1} in the mid-follicular phase and at 38.10(±0.2)°C and 14.6(±2.2) ml•100 ml^{-1}•min^{-1} in the mid-luteal phase. Both T_c and SkBF were significantly increased at the onset of vasoconstrictor activity in the luteal phase ($p \leq 0.05$). Heart rate averaged 150 (±11) and 161 (±9) beats•min^{-1} at the break point during the follicular and luteal phases respectively. These data indicate that onset of vasoconstrictor activity occurred at an increased T_c and SkBF during the luteal than in the follicular phase and therefore is not dependent on a critical exercise SkBF or T_c but may be explained by another regulatory mechanism, perhaps arterial pressure regulation.

Key words: Follicular phase, Luteal phase, Thermoregulation, Vasoconstrictor activity

Skin blood flow (SkBF) is linearly related to core temperature (T_c) above some threshold temperature at varying skin temperatures during exercise and at high skin temperatures during rest. However, SkBF cannot continue to increase infinitely while T_c continues to increase during exercise. Specifically, when skin temperature is high during moderate to severe exercise, there is a break point in the SkBF to T_c relationship such that at a T_c of approximately 38°C the increase in SkBF per unit increase in T_c is attenuated (3,19, 20, 23). Brengelmann et al. (3) interpreted this reduction in sensitivity at the higher level of T_c as vasoconstrictor activity resulting from arterial blood pressure regulation. The reduction in sensitivity was not the result of attaining a maximal SkBF, as local heating and supine rest at the same level of T_c caused much higher SkBF. Another laboratory reported that this "vasoconstrictor" response occurred at a SkBF above 15 ml • min^{-1} • 100 ml^{-1} and esophageal temperature (T_{es}) of approximately 38°C during semi-upright exercise (19). When exercise was done in the supine position the vasoconstrictor response was prevented. SkBF exceeded 26 ml • min^{-1} • 100 ml^{-1} and T_{es} reached 38.5°C (19). Roberts and Wenger (22) proposed that the reduction in SkBF during upright exercise in the heat could be mediated by a cardiopulmonary baroreflex as the decrement in SkBF was proportional to a decrease in stroke volume.

At least some of the attenuation of the SkBF response can be explained as a compensation for plasma volume shifted out of the vascular compartment (13,23). Rowell (23) has suggested that the break in SkBF and the decline in the rate of rise of cardiac output at a core temperature of 38°C (24) marks the point at which blood pressure regulation becomes dominant over the vasodilatory drive of increasing core temperature, therefore a relative vasoconstriction occurs.

One method to study the onset of vasoconstrictor activity at high levels of core temperature in individuals is to study women during both the follicular and luteal phases of the menstrual cycle. During the luteal phase, core temperature is elevated by approximately 0.3°C (17), thus two levels of core temperature can be evaluated. It has been observed that the T_{es} threshold for initiation of cutaneous vasodilation is increased (approximately 0.3°C) during exercise in the luteal phase (11, 12, 25). If the vasoconstrictor activity depended solely on a blood volume shift, then it might be hypothesized that SkBF would increase to a similar level in both phases of the menstrual cycle before the SKBF:T_{es} curve would be altered. In this case, the onset of the vasoconstrictor activity should occur at a higher core temperature during the luteal phase. However, plasma volume is decreased by approximately 200 ml in the mid-luteal phase compared to the mid-follicular phase (26, 29, 31) which when occurring singularly might result in a baroreflex onset at a lower SkBf than during the follicular phase. These conflicting phenomena of elevated core temperature and relatively lower plasma volume during the mid-luteal phase makes prediction of the effect of the menstrual cycle on the onset of the vasoconstrictor activity impossible. Therefore, this investigation was done to study how the menstrual cycle affects the onset of the vasoconstrictor activity at high internal temperatures during exercise.

Methods

Five healthy women (Table 1) volunteered to serve as subjects for a previously approved protocol. Each reported a normal menstrual cycle as defined

by a regular periodicity and had not used oral contraceptives for at least 6 months prior to the study. Daily basal body temperature (BBT) was taken during the course of the study to verify that a normal elevation in BBT (4) had occurred in the luteal phase. Luteal phase experiments were conducted when BBT was increased. Peak $\dot{V}O_2$ was determined before the study as described previously (18).

All experiments were conducted in the morning between 0730 and 1030 h and each subject was studied at the same time of day to eliminate circadian variation in thermoregulation (27). Each individual was studied in the follicular (days 3-8) and the luteal (days 21-24) phase. No food or drink except water was permitted after 2200 h the evening before the experiment.

Each subject reported to the laboratory dressed in shorts, a sleeveless shirt, shoes and socks. The environmental chamber was pre-heated to an ambient temperature (T_a) of 35°C, with an average ambient water vapor pressure of 1.73 kPa. The subject was weighed, then rested in the chair of a modified cycle ergometer (2). Each subject placed a catheter containing a thermocouple in her esophagus to the level of her heart for the measurement of core (esophageal) temperature (T_{es}) and was required to drink ~170 ml water while swallowing the T_{es} probe. Thermocouples were attached to the skin at eight sites to measure local skin temperatures which were used to calculate mean skin temperature (T_{sk}). All thermocouples were copper-constantan and were accurate to 0.1°C. T_{sk} was calculated from area weighting of each regional skin temperature (8, 21). using the equation:

$$T_{sk} = 0.07\ (T_{head}) + 0.175\ (T_{chest}) + 0.175\ (T_{back}) + 0.07\ (T_{upper\ arm}) + 0.07\ (T_{forearm}) + 0.05\ (T_{hand}) + 0.19\ (T_{thigh}) + 0.20\ (T_{calf})$$

ECG electrodes were attached to the chest for heart rate measurement in subjects 1-3 and heart rate was measured by auscultation using an automatic blood pressure monitor (Accutorr) in subjects 4 and 5. Venous occlusion plethysmography (14) was used to measure forearm blood flow (FBF). The measured FBF was used as an index of SkBF.

In three of the five (subjects 1-3) a catheter was placed in an arm vein so that multiple blood samples could be drawn during the experiment. Before the resting blood sample was drawn, each subject had been seated in the chair of the modified cycle ergometer for a thirty min period so that equilibration of body fluids was complete (9).

Resting data were collected for at least 20 min. T_{es}, $\bar{T}_{sk}$ and FBF were measured every 30 s. HR was measured every 5 min. Metabolic rate was estimated by an open circuit spirometry after 15 min of rest.

The subjects cycled at ~80% peak aerobic power for 35 min or until they could no longer maintain the workload. T_{es}, $\bar{T}_{sk}$, FBF and HR were measured as described for rest. Metabolic rate was measured at 20-25 min of exercise. Blood samples were drawn at 1, 3 and approximately 10 min of exercise and when the subjects' core temperature increased to 38°C. A fifth blood sample was drawn when one of the investigators observed a markedly reduced FBF and the last sample was drawn during the last two min of exercise.

Blood volume was estimated in subjects 1-3 by the method of Allen et al. (1). Plasma volume was calculated from the estimated blood volume and hematocrit with no correction for F-cell ratio. Hemoglobin concentration (Hb) was measured using an hemoglobinometer (Coulter Electronics). Hematocrit (Hct) was measured in triplicate by the microhematocrit technique. Relative

changes in plasma volume during the experiments were calculated from Hb and Hct (28).

The T_{es} threshold for the initiation of cutaneous vasodilation was calculated for each individual experiment by analyzing the exercise transient phase of the FBF:T_{es} relationship as described earlier (25). Linear regression equations were calculated to describe the SkBF:T_{es} relationship for each experiment during the initial rapid increase in both SkBF and T_{es} during exercise and when SkBF had become relatively attenuated while T_{es} continued to increase. The intersection of the two equations was defined as the "break point" in the SkBF:T_{es} relationship and was interpreted as the onset of vasoconstrictor activity. SkBF and T_{es} at that point were calculated for each experiment. A one way analysis of variance with repeated measures was used to test differences between the follicular and luteal phases for the T_{es} threshold for initiation of cutaneous vasodilation, the T_{es}, SkBF and heart rate at the onset of vasoconstrictor activity cycle, and time of exercise at the onset of the relative vasoconstriction. All data are reported as means ($\bar{X}$) and are presented with the standard deviation (S. D.). All differences are reported at $p \leq 0.05$, unless noted otherwise.

Results

During rest at 35°C the mean core temperature (T_{es}) was 36.83 ($\pm$ 0.2)°C during the follicular phase. In the luteal phase, mean resting T_{es} increased significantly to 37.13 ($\pm$ 0.2)°C. $\bar{T}_{sk}$ at rest was increased in the luteal phase (36.18 $\pm$ 0.3°C) as compared to the follicular phase (35.87 $\pm$ 0.3°C). Forearm blood flow and metabolic rate (M) were not significantly different between

menstrual cycle phases at rest (Table 2), although resting heart rate (HR) tended ($p = 0.07$) to be greater in the luteal phase (Table 2). Plasma volume averaged 3.1 ($\pm$0.1) ℓ in the follicular phase and 3.0 ($\pm$0.1) ℓ in the luteal phase for the three subjects studied and was significantly different between phases.

During exercise (approximately 80% peak $\dot{V}O_2$), the T_{es} threshold for onset of cutaneous vasodilation was significantly increased from 37.03 ($\pm$ 0.1)°C in the follicular phase to 37.37 ($\pm$ 0.2)°C in the luteal phase (Table 2), as has been reported previously (11, 12, 25).

At the time of the metabolic rate measurement (22 min of exercise) T_{es} was still increased in the luteal phase ($p = 0.056$) by an average 0.3°C (Table 3). $\bar{T}_{sk}$, M, HR, and FBF were not significantly different by the 22nd min of exercise.

The T_{es} for onset of vasoconstrictor activity as determined by the intersection of the two regression equations was significantly increased in the luteal phase and averaged 38.1 ($\pm$ 0.2)°C which was 0.58°C greater than during the follicular phase (Table 4). The FBF at the onset of vasoconstrictor activity averaged 10.9 ($\pm$2.4) and 14.6 ($\pm$ 2.2) $ml \cdot 100ml^{-1} \cdot min^{-1}$ in the follicular and luteal phases respectively. The onset of vasoconstrictor activity occurred earlier during exercise in the follicular phase than in the luteal phase (Table 4). Heart rate tended to be greater during the luteal phase at this break point ($p = 0.1$). In the three subjects from whom blood samples were drawn, PV was significantly lower in the luteal phase at the time of the relative vasoconstriction (Table 4). The PV decrease at the break point was approximately -18% in the follicular phase and -20% in the luteal phase.

Discussion

In a previous investigation (25) we described an increase in the T_{es} threshold for both sweating and cutaneous vasodilation during exercise in the luteal phase of the menstrual cycle. Other laboratories reported an increased threshold for onset of finger flood flow during exercise (12), and an increased cutaneous vasodilation, sweating and shivering threshold during passive heating or cooling in the luteal phase (10). Hessemer and Bruck (11) also reported an increased core temperature threshold for onset of vasodilation and sweating in the luteal phase during exercise in a cool environment in the early morning. These studies have made it clear that women who have the normal increase in basal body temperature (BBT) during the luteal phase have altered thermoregulatory control during the luteal phase. This concept is important to the understanding of the current investigation because the altered control of thermoregulation in the luteal phase was a means to study the "break point" in the SkBF:T_{es} relationship at two distinct levels of T_c in the individual women studied. In the current investigation, the resting T_{es} data (Table 2) and the increased T_{es} threshold for onset of cutaneous vasodilation in the luteal phase (Table 2) indicated that the women studied had the normal variation in thermoregulatory control during the menstrual cycles.

The increased resting $\bar{T}_{sk}$ observed in the luteal phase would be expected to shift the FBF:T_{es} curve slightly to the left as Wenger et al. (30) have shown that increased T_{sk} decreased the cutaneous vasodilatory threshold temperature. Yet, in the current study, the FBF:T_{es} curve was shifted to the right by approximately 0.3°C during the luteal phase despite the ~ 0.3°C increase in $\bar{T}_{sk}$.

Data from male subjects from the investigations by Brengelmann et al. (3) and Nadel et al. (19) are compared to the data from our females subjects in Table 6. The data designated by an asterisk (*) are mean values which were graphically extrapolated by the authors from raw data from 6 subjects presented by Brengelmann et al. (3). The exercise intensity used in the current study was 10% greater than the Nadel et al. study and was substantially greater than in the Brengelmann et al. study (3), as indicated by heart rate data. The duration of the exercise in the current study was slightly longer than in the other two studies. Brengelmann et al. used a water-perfused suit to control T_{sk} to 38.0°C, while T_{sk} in the other two studies was 2-2.5°C lower. The increased T_{sk} in the study of Brengelmann et al. (3) may explain the lower threshold for initiation of cutaneous vasodilation as compared to Nadel et al. (19) and the women in the current investigation during the follicular phase of the menstrual cycle. The vasodilatory threshold in the women was increased approximately 0.4°C in the luteal phase as compared to the men and the women in the follicular phase. The slope of the FBF:T_{es} relationship before the "break point" was similar in men (19) and women in the follicular phase although the slope was somewhat lower in men in the other study (3). In the luteal phase, the slope of the FBF:T_{es} was slightly increased, but this was not significantly different from the follicular phase (Table 5). In all three studies the gain of the FBF:T_{es} decreased significantly after the "break point" (by definition). However, the average slope after the break point was negative (increased T_{es} caused a decreased FBF) in the women. The T_{es} at the breakpoint averaged 38.0°C in the men in both studies (3, 19). When we subjectively determined the break point from individual data from one study (3), it ranged from 37.6-38.5°C. In

the follicular phase, the women had a lower T_{es} for onset of vasoconstrictor activity (37.53°C) than the men, but the T_{es} threshold was slightly higher (0.1°C) in the luteal phase than men. However, T_{es} at the onset of vasoconstrictor activity was significantly different between phases. The SkBF at the break point was greater in the luteal phase than the follicular phase and approximated that of the men in Nadel's study (19).

The negative slope after the break point, lower T_{es} and lower FBF at the break point in the women during the follicular phase than in men may be explained in part by the greater exercise intensity at which the women were working and by the longer exercise duration. Since relative sympathetic nervous activity is a function of $\dot{V}O_2$ max (5), there was probably a higher concentration of circulating norepinephrine (5,7) in the women and an increased PRA as well (6). Both NE and the product of renin, angiotensin II (AII), cause vasoconstriction.

Blood volume is reduced as a function of exercise intensity. The decrement in central blood volume may have contributed to the onset of the vasoconstrictor activity. At approximately the time of the break point in the FBF:T_{es}, PV had decreased by 18-20% in the women. Although Nadel et al. (19) did not report the PV at the break point in the FBF:T_{es} curve, they reported that PV decreased to 16.5% by approximately the end of exercise (20 min).

A relative vasoconstriction during exercise with increasing core temperature (break point in the FBF:T_{es} curve) has been interpreted as the result of blood pressure regulation at the expense of thermoregulation (3, 19, 20, 22, 23). Rowell (23) has written a comprehensive review describing the response of different vascular beds to exercise and heat stress. Briefly, cardiac output increases

substantially during exercise and roughly doubles during heat stress. Yet, demand continues for blood flow to the muscle to sustain exercise and to the skin for heat transfer. Eventually blood demands exceeds the cardiac output and CVP decreases, initiating a cardiopulmonary baroreflex. Regional blood flow is compromised at specific sites, one of which is the skin, to maintain CVP (23). Decreased central blood volume and increased cutaneous venous volume both decrease CVP (24). In this study (n = 3), PV was slightly lower at the break point in the luteal phase than in the follicular phase. Apparently the slightly decreased PV in the luteal phase was not sufficient to result in an earlier onset of vasoconstrictor activity. Furthermore, FBF was increased at the break point in the luteal phase even though PV was decreased.

The observation that SkBF increased at the break point in the luteal phase is not surprising as both T_{es} and T_{sk} were increased in the luteal phase at rest (Table 2) and SkBF normally increases an a function of increasing T_{es} and T_{sk}. On the other hand, an elevated T_{sk} could also result in a more capacious venous component of the cutaneous vascular bed. If that were true the onset of vasoconstrictor activity would theoretically occur more quickly in the luteal phase to overcome a lower CVP resulting from the increased blood transit time in the veins.

There is an increased concentration of PRA both at rest (15, 26) and during exercise (26) in the luteal phase as compared to the follicular phase which suggests different sympathetic outflow to the kidney. Since increased PRA is an index of the production of the vasoconstricting agent AII, it would be expected that the onset of vasoconstrictor activity would occur more quickly in the luteal phase when PRA is elevated. Perhaps the increased PRA measured

in other studies (15, 26) at rest and during steady state exercise is the result of decreased responsiveness of the cutaneous vasculature in the luteal phase.

In conclusion, we have observed an attenuation of the SkBF:T_{es} relationship in women exercising at 80% peak $\dot{V}O_2$ such that there is a smaller increase in SkBF per unit increase in T_{es} after approximately 8 min of exercise in the follicular phase and after 15 min in the luteal phase. The reason for the relative delay in the onset of vasoconstrictor activity in the luteal phase is unknown and further studies are necessary to investigate whether this delay is due to reduced responsiveness of the cutaneous vascular bed, reduced sympathetic nervous activity to the cutaneous vasculature or some other mechanism.

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Table 1. Individual subject characteristics

Subject	Height (cm)	Weight (kg)	Age (yr)	$\dot{V}O_2$ peak ($\ell \cdot min^{-1}$)	Workload ($\%\dot{V}O_2$)	A_D[1] (m^2)
1	173	64.0	33	2.70	84	1.76
2	163	65.9	22	2.64	82	1.71
3	170	60.0	31	2.55	83	1.69
4	163	61.7	26	2.65	77	1.66
5	173	77.8	26	3.83	71	1.92
$\bar{X}$	168.4	65.9	27.6	2.87	79.4	1.75
S.D.	5.08	7.0	4.4	0.54	0.10	0.10

[1] DuBois body surface area

Table 2. The resting forearm blood flow (FBF), esophageal temperature (T_{es}), mean skin temperature (T_{sk}), metabolic rate (M), heart rate (HR) and the T_{es} threshold for onset of cutaneous vasodilation (threshold).

Subject	FBF	T_{es}	$\bar{T}_{sk}$	M	HR	Threshold
	(ml•100 ml•min^{-1})	(°C)	(°C)	(W•m^{-2})	(b•min^{-1})	(°C)
Follicular Phase						
1	6.6	37.03	36.26	45	68	37.24
2	3.2	36.71	35.72	44	65	36.96
3	3.4	37.07	35.70	41	68	37.02
4	2.1	36.56	35.62	37	67	36.92
5	6.9	36.82	36.03	59	64	37.02
$\bar{X}$	4.4	36.84	35.87	45.2	66.8	37.03
S.D.	(2.2)	(0.21)	(0.27)	(8.3)	(1.6)	(0.1)
Luteal Phase						
1	6.5	37.26	36.57	41	70	37.52
2	5.5	36.96	36.23	42	81	37.29
3	3.7	37.35	35.98	43	68	37.58
4	4.0	36.85	35.87	49	72	37.20
5	3.8	37.25	36.24	65	75	37.28
$\bar{X}$	4.5	37.13*	36.18*	48.0	73.2	37.37*
S.D.	(1.5)	(0.22)	(0.27)	(10.0)	(5.1)	(0.2)

An asterisk (*) indicates differences between follicular and luteal phases ($p \leq 0.05$).

Table 3. Forearm blood flow (FBF), esophageal temperature (T_{es}), mean skin temperature (T_{sk}) and metabolic rate (M) at 22 min of exercise.

Subject	FBF	T_{es}	$\bar{T}_{sk}$	M	HR
	($ml \cdot 100ml^{-1} \cdot min^{-1}$)	(^oC)	(^oC)	($W \cdot m^{-2}$)	($b \cdot min^{-1}$)
Follicular Phase					
1	13.3	37.96	34.63	404	150
2	9.5	38.00	35.55	416	166
3	10.8	38.08	35.01	395	167
4	7.4	37.86	34.35	424	149
5	15.2	38.76	35.86	516	166
$\bar{X}$	11.2	38.13	35.08	431	160
S.D.	(3.1)	(0.36)	(0.63)	(49)	(9)
Luteal Phase					
1	11.6	38.31	34.75	417	150
2	13.9	38.04	35.55	455	167
3	11.3	38.77	34.84	447	162
4	9.5	38.19	36.34	484	148
5	17.9	38.87	35.96	507	168
$\bar{X}$	12.8	38.44*	35.17	462	159
S.D.	(3.2)	(0.37)	(0.55)	(35)	(9)

An asterisk (*) indicates differences between the follicular and luteal phases ($p \leq 0.05$).

Table 4. The esophageal temperature (T_{es}), mean skin temperature ($\bar{T}_{sk}$) forearm blood flow (FBF), time, plasma volume, change in plasma volume (ΔPV) and heart rate (HR) at the onset of vasoconstrictor activity.

Subject	T_{es}	$\bar{T}_{sk}$	FBF	Time	PV	ΔPV	HR
	(°C)	(°C)	(ml•100 ml^{-1}•min^{-1})	(min)	(ℓ)	(%)	(b•min^{-1})
Follicular Phase							
1	37.60	35.33	13.7	6.5	2.57	-19.93	150
2	37.59	35.33	9.4	12.5	2.50	-17.60	166
3	37.40	35.38	8.2	5.0	2.54	-16.81	143
4	37.37	34.56	10.2	10.0	-[a]	-	149
5	37.68	35.67	13.2	7.5	-	-	149
$\bar{X}$	37.53	35.25	10.9	8.3	2.54	-18.1	150
S.D.	(0.14)	(0.41)	(2.4)	(3.0)	(.04)	(1.6)	(11)
Luteal Phase							
1	38.10	34.99	15.4	11.5	2.39	-23.15	154
2	37.80	35.49	15.8	15.0	2.39	-18.64	172
3	38.20	35.68	13.2	12.5	2.41	-18.91	165
4	38.20	34.91	11.6	26.0	-	-	150
5	38.19	35.96	17.1	11.0	-	-	163
$\bar{X}$	38.10*	35.41	14.6*	14.8*	2.40*	-20.2	161
S.D.	(0.17)	(0.45)	(2.2)	(5.4)	(.01)	(2.5)	(9)

An asterisk (*) indicates differences between the follicular and the luteal phases ($p \leq 0.05$)

[a] Blood samples were obtained in subjects 1-3 only.

Table 5. The individual slopes and y-intercept generated from linear regression equations for the forearm blood flow to esophageal temperature relationship for the initial exercise transient phase and after relative vasoconstriction occurred.

	Exercise Transient		Relative Vasoconstriction	
	Slope	y-Intercept	Slope	y-Intercept
		Follicular Phase		
1	19.73	-728.1	-4.60	186.7
2	9.57	-350.5	-0.60	32.0
3	10.67	-390.9	1.00	-29.0
4	16.79	-617.3	-3.60	144.7
5	13.33	-490.5	-2.19	98.5
6	9.47	-343.7	1.09	-27.9
		Luteal Phase		
1	16.60	-616.3	-8.82	351.2
2	21.09	-781.0	1.56	-43.2
3	15.07	-562.3	-3.68	153.8
4	16.52	-610.5	2.53	-86.1
5	12.28	-459.9	3.89	-138.0
6	15.80	-586.3	-0.98	54.5

Table 6. The exercise intensity, duration, mean skin temperature (T_{sk}), vasodilatory threshold, slope of the FBF:T_{es} curve before the breakpoint, slope of the FBF:T_{es} after the breakpoint, T_{es} at the breakpoint, and FBF at the breakpoint for men (Brengelmann et al. (3) and Nadel et al. (19) and women in the follicular (F) and luteal (L) phases.

	Males (3)	Males (19)	Females	
			Follicular	Luteal
Exercise intensity	86-147W	70% $\dot{V}O_2max$	80% $\dot{V}O_2max$	80% $\dot{V}O_2max$
Duration (min)	17-30	20-25	30-35	30-35
T_{sk} (°C)	38.0	35.5	35.9	36.2
Vasodilatory threshold (°C)	36.84*	36.95	37.03	37.37
Slope pre-break ($ml \cdot 100ml^{-1} \cdot min^{-1} \cdot °C^{-1}$)	7.6	13.1	13.3	16.2
Slope post-break ($ml \cdot 100ml^{-1} \cdot min^{-1} \cdot °C^{-1}$)	2.12	4.8	-1.48	-0.92
T_{es} at break (°C)	38.0	38.0	37.53	38.10
FBF at break ($ml \cdot 100ml^{-1} \cdot min^{-1}$)	12.48*	15.1	11.2	14.6

An asterisk (*) denotes mean values calculated from data presented by Brengelmann et al. (3).

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